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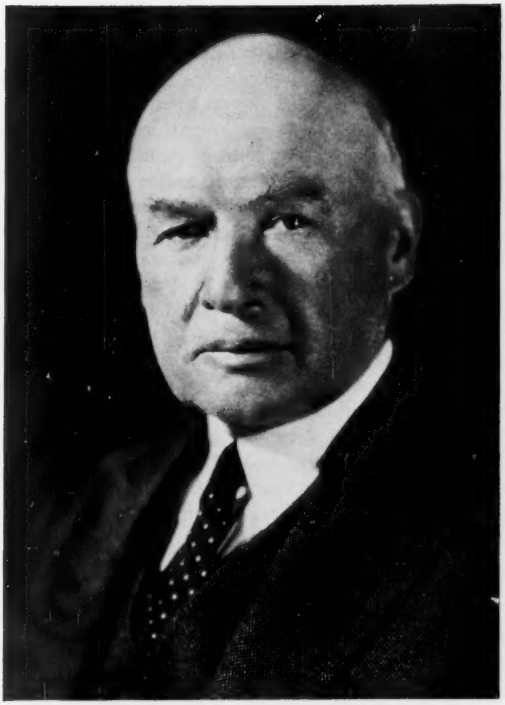

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"MECHANICAL BACKACHE"—THE MOST COMMON TYPE OF LOW BACK PAIN

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BECAUSE low back pain is so commonly encountered, the physician must learn to recognize accurately the various causes of backache and to institute the proper treatment himself or direct the patient to another physician who is capable of carrying out the necessary treatment. The physician's lack of interest in this important problem accounts for the prevalence of various medical cults which specialize in the treatment of back pain.

To maintain the erect position, one must have good body balance. This balance may be upset by the presence of certain mechanical factors such as faulty posture, obesity, relaxed abdominal musculature, weak back musculature, and inequality of length of the lower extremities. When backache is caused by some disturbance in the normal body balance through the action of one or more of these mechanical factors, the condition is called a "mechanical backache." The same mechanical factors often play a significant role in producing at least a portion of the low back pain associated with fibrositis, hypertrophic arthritis, rheumatoid spondylitis, spondylolisthesis and other causes of backache. Nevertheless the term "mechanical backache" should be reserved for the case in which there is no demonstrable pathologic condition to account for the pain within the back or pelvis other than mechanical factors.

Good posture is essential to good body balance. When a person stands correctly, a perpendicular line dropped from the ear should pass through the shoulders, hips and ankle joints (fig. 1a). With good posture, the head is high, chin up and in, chest up, shoulders relaxed and back, abdomen flat, lower back flattened, knees straight, and feet parallel. Little actual muscle energy is required to maintain the erect position with good posture because the body is so rightly balanced. It is easy to see, however, how quickly normal body balance is upset by a sagging abdomen, a flattened chest, humped shoulders and a swayed back (fig. 1b). With poor posture, unnatural stresses and strains are thrown upon the muscles and ligaments which support the vertebral column, and these stresses and strains become most pronounced at the lumbosacral joint. Discrepancy in length of the lower extremities of one-half inch or more may also cause sufficient tilting of the pelvis to produce abnormal strain of the back musculature. A normal pelvis may compensate satisfactorily for a shortening of as much as one and one-half inches in one leg; but a greater discrepancy in leg length cannot be counteracted by pelvic tilt alone without causing scoliosis. Unless the patient wears on the shoe of the short leg an elevation of sufficient height to reduce the discrepancy to a value comfortably equalized by lateral tilting of the pelvis, pain in the lower back may result. It is unusual for a discrepancy in leg length of one-half inch or less, to cause pain in the lower back because of strain on back musculature.

"Mechanical backache" is much more frequently encountered in women

than in men. This is because of the prevalence among women of obesity, poor posture, and weak musculature. Low back pain may be the presenting symptom in cases of chronic nervous exhaustion. This condition, also, is more common in women than in men.

Diagnosis

The diagnosis of a "mechanical backache" should be made only after carefully evaluating the patient's history, physical condition and roentgenograms of the lumbo-sacral spine. The physician cannot omit any one of these three items. This diagnosis indicates that no actual disease or permanent structural abnormality is responsible for the backache.

The usual history is one of a gradual onset of low back pain. This may be well localized to the lumbo-sacral region, but often the patient complains of diffuse discomfort across the entire lower back extending into the buttocks; there is no radiation of the pain into the lower extremities. The back pain is usually of many years' duration, although the patient is infrequently incapacitated since the pain is seldom severe. The attacks of pain are more likely to be an acute muscular and ligamentous strain superimposed upon, and perhaps precipitated by, the factors producing a "mechanical backache."

The pain of a "mechanical backache" is aggravated by activity and relieved by rest. Standing or sitting for lengthy periods may increase the discomfort. Changes in weather have no effect on the patient's symptoms; coughing, sneezing and straining have little effect on the pain.

While undergoing examination, the patient should be completely disrobed, or partially covered so that an adequate view of the entire back may be obtained. Usual factors contributing to "mechanical backache" are faulty posture with increased lumbar lordosis and increased rounded dorsal kyphos. There often exist varying degrees of obesity with a prominent sagging abdominal panniculus. Corpulence is not an essential diagnostic criterion, however, since the tall, thin person sometimes has faulty posture with weak back and abdominal musculature. There is often localized tenderness in the region of the lumbo-sacral joint and over the iliolumbar ligaments. Movements of the back may or may not be restricted; limitation of back motion is not an essential physical finding. There is a normal range of hip motion. Raising the body from the supine to the sitting position without support of the arms is often difficult, depending upon the degree of weakness of back and abdominal musculature. There is no evidence of any peripheral nerve irritation; knee and ankle reflexes are equal and active. The "straight leg raising" test is negative; that is, with the patient supine, the lower extremity can be elevated passively, with knee completely extended, to a position of almost 90 degrees without undue discomfort. Should there be discomfort associated with this test it occurs in the hamstring tendons back of the knee due to the normal tightening of these structures. The legs are measured from the anterior superior iliac spine to the medial malleolus. A discrepancy of one-half inch or more in leg length is possibly significant in causing low back pain.

Roentgenograms of the lumbo-sacral spine should be studied carefully with particular attention to the presence of arthritic changes or structural abnor-

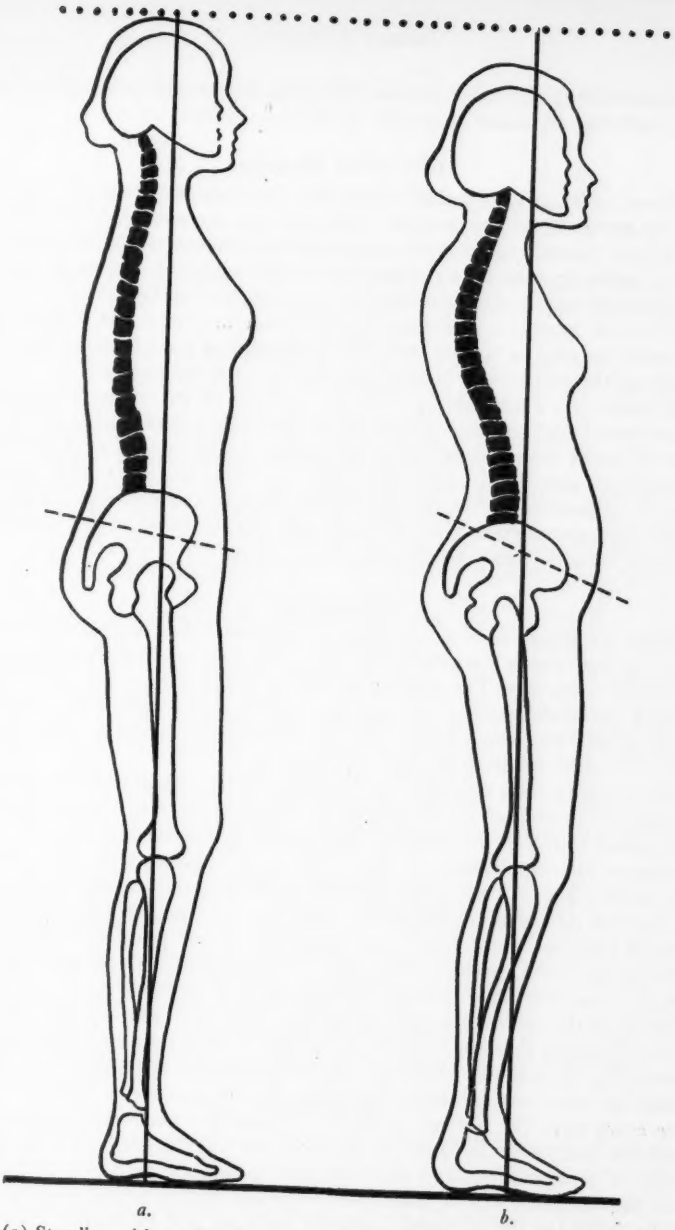

FIG. 1. (a) Standing with good posture, a perpendicular line dropped from the ear passes through the shoulders, hips and ankle joints. (b) Normal body balance is upset by a sagging abdomen, a flattened chest, humped shoulders and a swayed back, producing a "mechanical backache."

malities involving the lower lumbar spine. In the simple "mechanical backache," nothing abnormal is noted.

Differential Diagnosis

Chronic fibrositis, so-called "muscular rheumatism," is often associated with the syndrome of "mechanical backache." In these cases there frequently are "trigger points," or localized areas of severe tenderness. Patients also complain of stiffness in the back muscles following a period of inactivity; this stiffness is usually most severe on arising and subsides during the day.

An acute muscular and ligamentous strain involving the lumbar spine may easily become a chronic one if the original injury is neglected and if one or more of the mechanical factors described are already present.

In cases of rheumatoid spondylitis, motions of the spine are restricted oftener than in a "mechanical backache." The Marie-Strümpell type of spondylitis is found more frequently in men than in women and is characterized by tenderness over the spine, limited chest expansion, and the typical roentgenographic appearance of sacro-iliac calcification in the paraspinal ligaments.

The roentgenograms also serve to differentiate between a "mechanical backache" and a backache due to other lesions.

Treatment

Once the diagnosis of a "mechanical backache" has been established, the physician may assure his patient that the condition may be alleviated by certain simple measures. The patient should be urged to sleep on a firm bed. A piece of plywood between the mattress and springs is advisable, and a stiff cotton or felt mattress is better than an innerspring mattress. This firm bed prevents further strain to the back musculature.

Heat aids in the relaxation of the back muscles and stimulates circulation. The patient is advised to take a hot tub bath nightly, remaining in the water for a period of five to ten minutes. This should be followed by twenty to thirty minutes of heat application on the lower back, employing an infra-red lamp or a heating pad. A brief gentle massage applied to the patient's back is helpful, but not an essential part of the treatment. The patient then performs the series of exercises which have been prescribed by the physician and in which he or she has been carefully instructed. It is not enough to advise a patient to take exercises. He must be shown exactly how to perform them, and the description of the exercises should be written down and given to the patient for ready reference. Too much exercise is unwise at first because of the danger of over-taxing the already weakened back musculature. If possible, the exercises should be done both morning and night. It is imperative to do them at least once every day. The patient should be advised that little improvement in the condition can be anticipated for at least two months, since it will take this period of time to strengthen the back musculature satisfactorily. In most cases some improvement will be noted before the two month period has elapsed.

The following list of simple postural exercises will be found adequate for most cases of "mechanical backache." If possible, each exercise should be done

five times at the onset and gradually increased over a period of several weeks or months to ten or fifteen times, depending upon the exercise tolerance of the individual patient.

A. Lie on back with knees bent:
   1. Tilt the pelvis, flattening the lower back against the floor.
   2. With pelvis tilted, raise the head and shoulders from the floor.
   3. With pelvis tilted, raise legs alternately.
   4. With pelvis tilted, straighten out one leg and then the other.

B. Stand with heels four to six inches from the wall and with back against the wall:
   1. Tilt the pelvis, flattening the lower back against the wall.
   2. With back flat, leave the wall; come up and down on toes; return to wall.
   3. With back flat against the wall, raise legs alternately.
   4. With back flat, walk away from the wall.

If more vigorous exercises are indicated to strengthen the back and abdominal muscles, the following may be prescribed:

1. Lie on back, elevate both legs with knees completely extended.
2. Lie on back with arms overhead, rise to sitting position and touch toes.
3. Lie on abdomen with arms at side, raise head and shoulders, arching back.

Physical therapy treatments—diathermy or radiant heat and massage—may be administered several times a week, but are not essential. A corset is recommended only if the patient is quite obese and the abdominal musculature especially relaxed. In "mechanical backache," a corset must be considered a temporary relief measure, to be discarded when the back becomes strong enough to get along without it. The wearing of a corset for a long period of time delays the permanent cure, although the immediate effect may be beneficial.

General systemic measures may be an important adjunct in the treatment of the "mechanical backache." Vitamin supplements may be indicated, a weight reduction diet may be needed, and a mild sedative or a tonic may be prescribed by the physician in a particular case. Proper treatment must also be instituted to control any other disease which may be present, such as chronic cholecystitis, peptic ulcer, or an irritable bowel. It is important to tell the patients, however, that such treatment is directed toward improving their general health and is not specifically prescribed for the cure of the backache.

Conclusion

The "mechanical backache" is the most common type of low back pain. Because the condition is seldom incapacitating, the physician is apt to minimize its importance. Once the diagnosis has been made by careful examination, an adequate program of treatment should be promptly instituted.

HEREDITARY PSEUDOHEMOPHILIA

Report of 4 Cases

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ABNORMAL bleeding is a frequent clinical problem, and as a rule its diagnosis offers the clinician little difficulty. In the majority of patients with hemorrhagic diathesis the bleeding is associated with (1) an altered permeability of the capillary endothelium, allowing the escape of erythrocytes from the blood vessels, or (2) an abnormality of the clotting mechanism itself. Increased vascular permeability may be due to a platelet deficiency, as in thrombocytopenic purpura, or to impairment of the endothelial nutrition, as in vitamin C deficit. Various drugs, toxins, and possibly certain allergic states are also responsible for capillary injury and increased permeability capable of producing abnormal bleeding. Hemorrhagic diseases such as hemophilia, hypothrombinemia, and afibrinogenemia are associated with a defective clotting system.

An atypical variety of hemorrhagic diathesis occurs, however, which does not conform to the entities embraced by the usual classification of bleeding problems. Known as hereditary pseudohemophilia, this disease is characterized by excessive bleeding associated with prolonged bleeding time, normal platelets, without demonstrable defect in the clotting mechanism, and normal clot retraction.

Case Reports

Case 1. A boy, aged 6, was referred because of abnormal bleeding since infancy. He had bled profusely from minor wounds, and several times slight cuts had produced bleeding which lasted for several hours. At the age of 3 he had fallen, causing severe bleeding from the throat and mouth for twenty-four hours. This had necessitated hospitalization for four days. After this episode he was apparently asymptomatic until three weeks before admission, when he developed a sore throat which lasted for one week and was followed by continuous oozing and bleeding from the oral cavity for two weeks. For three days prior to admission the patient had epistaxis.

The past medical history was negative and he was otherwise in perfect health. Family history disclosed that the mother was a "bleeder," but other cases of bleeding in the family could not be discovered.

On physical examination the blood pressure was 88/68 and the temperature 98.6 F. There were no petechiae; however, the tourniquet test was positive. There was some dried blood present in the left nostril and several areas of petechiae over the soft palate. A small hematoma was noted on the gingival margin in the left upper molar region. There was a soft pulmonic systolic murmur. The liver and spleen were not palpable, and there was no lymphadenopathy.

Examination of the blood on June 13, 1939, revealed 9.5 Gm. of hemoglobin (Haden-Hausser) per 100 cc., 3,860,000 erythrocytes, 8750 leukocytes, and 450,000 platelets per cu. mm.; bleeding time (Duke method) was more than thirty minutes; coagulation time (Lee-White) was fifteen minutes and prothrombin time (Quick) seventeen seconds (normal

seventeen seconds). The clot retraction was normal. Examination of the blood smears revealed a normal differential count and no abnormal cells. The blood Wassermann and Kahn tests were negative.

For the next month the patient received injections of Lederle's moccasin venom, 0.5 cc. every four days, and no bleeding occurred during this time. One year later the patient felt well but was again having excessive bruising and bleeding from small cuts. In March and May, 1943, he bled from the left tonsil, and in both instances the bleeding responded to several injections of snake venom. On February 20, 1946, the patient slipped on the icy sidewalk and his left leg doubled underneath him. He developed a large ecchymotic area about the left thigh with soft tissue swelling. A roentgenogram of the leg showed no fracture, but soft tissue calcification was noted and thought to be the result of an old hemorrhage.

The blood studies on February 20, 1946, approximately eight years after his first visit revealed the hemoglobin to be 9.8 Gm. (Haden-Hausser) per 100 cc., and there were 4,100,000 erythrocytes, 7900 leukocytes and 720,000 platelets per cubic millimeter. The bleeding time (Ivy method) was stopped after twenty-five minutes, and the coagulation time (four test-tube method) was ten minutes. Clot retraction was normal, but the clot was more friable than usual.

Case 2. A woman, aged 37, mother of the first patient, submitted to an examination at our request. Though she had always regarded herself as a "bleeder" she had never sought medical advice. As a child she had suffered from severe nose bleeds, particularly about the ages of 6 and 7 and during severe colds. She had had two permanent teeth removed and the bleeding was extreme on each occasion. She bruised easily and her gums bled freely even on slight trauma. Menstruation had always been normal and she had had abnormal vaginal bleeding on only two occasions. Six years before admission and twelve days following the birth of her only son, she had developed severe vaginal bleeding. In spite of two weeks of futile effort by the attending physician to stem the blood flow, it continued and became alarming. A hysterectomy was performed without difficulty. There was no subsequent vaginal bleeding, and the incision healed without undue oozing. She had continued to bruise easily. Past history was irrelevant. No history of familial hemorrhagic disease could be elicited.

On physical examination the blood pressure was 130/80 and the pulse 72. There were numerous bruises over her entire body, but the tourniquet test was negative. The liver and spleen were not palpable, and there was no lymphadenopathy.

Blood and hemorrhagic studies at this time disclosed 12.5 Gm. of hemoglobin (Haden-Hausser) per 100 cc., 4,610,000 erythrocytes, 7050 leukocytes, and 230,000 platelets per cubic millimeter. The bleeding time was more than one hour and forty-five minutes, the coagulation time was eleven minutes (Lee-White) and the prothrombin time (Quick) was fourteen seconds (normal nineteen seconds). The clot retracted normally. Examination of the blood smear was negative.

Case 3. A boy, aged 3, was admitted to the Cleveland Clinic Hospital on June 6, 1940, with severe epistaxis of six days' duration. He had bled to the point of extreme danger, and in spite of two blood transfusions the bleeding had remained uncontrollable. The child had experienced frequent easy bruising since infancy, and he had always bled excessively from small cuts. One year previously he had bled for twenty-four hours from a small cut on his tongue. In 1939 the patient had epistaxes lasting many days, but never with the vigor of his present one. Hemoptysis, hematuria, melena, or hemarthrosis had never been noted. However, he had vomited "almost a quart of blood" soon after onset of the present illness. He was otherwise in good health. Family history revealed that the patient's father was a "bleeder," and one of his great uncles on the paternal side was reported to have died of hemorrhage from a wound on his thigh at the age of 17.

The physical examination disclosed an acutely ill, pale, and exsanguinated child with rapid respiration. The temperature was 99 F. and pulse 124. The blood was dropping freely from both nostrils, and there were numerous dime-sized purpuric spots on the arms and legs. The tourniquet test was negative. A soft apical systolic murmur was present. The liver and spleen were not palpable, and there was no lymphadenopathy.

An examination of the blood on June 5, 1940 revealed 4.7 Gm. of hemoglobin (Haden-

Hausser) per 100 cc., 1,730,000 erythrocytes, and 450,000 platelets per cu. mm. The bleeding time (Duke method) was thirteen minutes, coagulation time (Lee-White) was five minutes; prothrombin time (Quick) was twenty seconds (normal nineteen seconds). Clot retraction was normal. The blood smear was negative. The blood Wassermann and Kahn tests were negative. Calcium was 9.3 mg. and plasma fibrinogen 269 mg. per 100 cc. of blood.

The child was admitted directly to the hospital and was given immediate transfusion. Bleeding decreased rapidly, and by the next day it had completely subsided. He was discharged after the third day and was placed on calcium carbonate, vitamin C, and occasional injections of snake venom. On October 10, 1940, the patient returned. He was continuing to have occasional epistaxis. Blood examination disclosed 10.0 Gm. hemoglobin (Haden-Hausser) per 100 cc., 10,650 leukocytes, and 500,000 platelets per cu. mm. The bleeding time (Duke) was twenty-eight minutes, clotting time (Lee-White) seven minutes, and there was normal syneresis.

The patient was not again heard of until recently, when through correspondence with his father we learned that the child had died on October 20, 1944. He had grown progressively worse and had had frequent nose bleeds, each more severe. He had developed hemarthrosis in both knees. He died following an episode of uncontrollable nasal hemorrhage that failed to respond to five blood transfusions.

Case 4. A man, aged 44, father of case 3, was examined at our request. He gave a history of severe nasal hemorrhages in earlier years. In 1921 he had had epistaxis "almost continuously" for sixteen days. He had experienced similar episodes throughout his entire childhood and at times had bled until he was quite pale and extremely weak. The epistaxis was unrelated to trauma and often began at night while he was asleep. For the past twenty-five years there had been bleeding from the gums, which upon two occasions was excessive, and which was so easily provoked that he had been unable to brush his teeth for many years. He had not had any extractions. There had been no hemoptysis, hematuria, melena, or hematemesis. He had one child aged 3 (case 3) who was a "bleeder" and another, aged 6, who was normal. One uncle on the maternal side had died at the age of 17 following uncontrollable hemorrhage from a wound on his thigh.

Upon physical examination his temperature was 99 F., pulse 84, and blood pressure 108/72. There was some bleeding from the gums and poor oral hygiene with fetor and pyorrhea. The heart had a grade 2 basal systolic murmur. The tourniquet test was negative. The spleen and liver were not palpable, and there was no lymphadenopathy.

Examination of the blood revealed 13 Gm. of hemoglobin (Haden-Hausser) per 100 cc., 4,890,000 erythrocytes, 5050 leukocytes, and 320,000 platelets per cubic millimeter. The bleeding time (Duke) was one hour and ten minutes; clotting time (Lee-White) was seven minutes. The clots retracted normally. Prothrombin time (Quick) was nineteen seconds (normal nineteen seconds). Fasting blood sugar was 106 mg. per cent. The serum calcium was 10.1 and plasma fibrinogen 292 mg. per 100 cc. The blood Wassermann and Kahn tests were negative. The blood vitamin C was 0.30 and 0.40 mg. per 100 cc.

On November 29, 1946, this patient was continuing to bleed from his gums and to bruise easily. Bleeding time (Ivy method) was still prolonged, being eighty-eight minutes; clotting time (four test-tube method) was eighteen minutes, and prothrombin time (Quick) was fourteen seconds (normal fifteen seconds). The hemoglobin (Haden-Hausser) was 11.5 Gm. per 100 cc.

Pathogenesis

The true pathogenesis of hereditary pseudohemophilia remains obscure. Glanzmann,[1] when describing his patients, speculated upon a functional deficiency in the platelets themselves as a basis for the prolonged bleeding. It was he who first named this condition "hereditary hemorrhagic thrombasthenia." Morawitz and Jürgens[2] and von Willebrand and Jürgens,[3] using an apparatus

called a capillarthrombometer, claim to have shown that the platelets in such cases do not agglutinate as rapidly as normal ones. They postulated that the disease was due to some qualitative defect in the platelets which prevented their agglutination within a normal time. Buckman,[4] however, was unable to demonstrate any abnormality in the blood platelets and found that the addition of a suspension of platelets from a patient with hereditary pseudohemophilia to hemophilic blood caused as rapid coagulation as a suspension of platelets from a normal person. Since the majority of these cases exhibit normal syneresis, it seems reasonable to conclude that the defect responsible for the abnormal bleeding may be elsewhere than in the platelets.

By means of direct visualization of the capillaries in the nail bed, MacFarlane[5] demonstrated a capillary defect in patients with pseudohemophilia. He noted the response of a normal capillary when injured by means of a glass fibre, and he compared the normal reactors with 5 cases of pseudohemophilia. He found that the platelets were of normal morphology in all 5 cases, and they could be visualized clumping actively in contact with the glass. The capillaries in the patients with pseudohemophilia, however, were distorted and displayed bizarre forms, and they did not constrict normally after injury in any instance. Perkins,[6] also studying the capillary loops in the human nail bed, was unable to demonstrate constriction of these vessels when subjected to trauma. These authors concluded that there is a functional defect in the capillaries themselves which is responsible for the excessive bleeding.

Symptomatology

Clinically, patients with pseudohemophilia are indistinguishable from those with thrombocytopenia. The bleeding tendency is as pronounced, and fatalities occur. One of our patients died from uncontrollable hemorrhage. The condition is usually noted in infancy and may be present at birth. It occurs in both sexes with almost equal frequency, being slightly greater in women in the small series so far reported. The age incidence of pathologic bleeding extends from infancy to old age, but as a general rule, as Minot[7] has pointed out, the severity tends to diminish as the patient grows older.

The paramount symptom of pseudohemophilia is the tendency to bleed severely from minor wounds. Epistaxis is present in practically all cases. Easy bruisability and the formation of large ecchymoses, sometimes spontaneously but more often post-traumatically, are next in frequency. Petechiae appear quite rarely. Bleeding from the mucous membranes is often present, especially hemorrhage from the gums and following tooth extractions. Frequently these patients are unable to brush their teeth because of the bleeding that ensues. Recurrent hemorrhage from the gastrointestinal tract, uterus, and kidneys has been reported. Hemarthrosis occurred in 1 of our patients, though its occurrence may be considered a rarity. The course of the disease is characterized by periods of apparent remission from symptoms, and there is no correlation between the bleeding time and the clinical severity.

Diagnosis

The principal and only consistently abnormal hematologic feature of pseudohemophilia is the prolonged bleeding time. This is an extremely variable factor, however, and it fluctuates in any given case from nearly normal to greatly prolonged. The coagulation time is normal, the platelets are normal and in some cases are increased. The prothrombin time, plasma fibrinogen, and blood calcium are not altered. The clot retraction is consistently normal in pseudohemophilia. The tourniquet test (Rumpel-Leede) is variable, being mildly positive in less than half of the cases and much less frequently positive than in true thrombocytopenia.

The disease must be differentiated from true hemophilia, in which the tendency to hemorrhage is accompanied by a prolonged coagulation time and a normal bleeding time, the antithesis of pseudohemophilia. The coagulation time of recalcified plasma, a test considered pathognomonic for hemophilia, is prolonged in that disease and normal in pseudohemophilia. In addition the characteristic hereditary pattern of hemophilia serves to distinguish the two diseases. The thrombocytopenic purpuras must also be excluded before the diagnosis of pseudohemophilia can be made. In this disorder the tendency to bleed is associated with a pronounced decrease in the number of circulating blood platelets which are normal in pseudohemophilia. The bleeding is usually of a chronic nature, though it may be acute. In thrombocytopenia the coagulation time is normal, the bleeding time prolonged, and the clot retraction poor or absent. The tourniquet test (Rumpel-Leede) is almost always positive in thrombocytopenic purpura. Hereditary pseudohemophilia may be distinguished from the so-called vascular purpuras by the presence of petechiae, a positive tourniquet test, and a normal bleeding time in the latter disease.

Treatment

The treatment of this disease is nonspecific and generally unsatisfactory. Various measures such as large doses of calcium, vitamin K, and ascorbic acid have been employed and have proved ineffectual. No real improvement has been obtained by the use of moccasin venom. Since the spleen is not involved in the pathogenesis of this disease, splenectomy is not indicated and may indeed be hazardous. Blood transfusions are useful in combating the anemia, but they are of little value in controlling the hemorrhage itself. Treatment with pressure bandages applied locally and the application of topical thrombin and fibrin foam are the most helpful measures of controlling bleeding.

Prognosis

Though the prognosis is exceedingly good in patients with pseudohemophilia, it is important to recognize the presence of the disease and to be cognizant of the fact that certain serious consequences may follow surgical procedures. Unlike hemophilia, in which blood transfusions or plasma will shorten

the coagulation time and control hemorrhage, or thrombocytopenic purpura in which splenectomy assures control of bleeding in most cases, there is no successful method of combating hemorrhage in pseudohemophilia. Patients, therefore, should not be subjected to elective operation.

Discussion

Though Glanzmann[1] in 1918 has been credited as the first to recognize this atypical hemorrhagic diathesis as a disease entity, it is doubtful that his cases represent true examples of pseudohemophilia. His patients were unusual in that they had a normal coagulation time and a normal platelet count, with the defect lying in the retraction of the clot, which was abnormal or completely absent, rather than in a prolonged bleeding time, which was normal. It is more likely that von Willebrand[8] in 1926 should be considered the first to have described this unusual form of bleeding, since he reported it among 16 women and 7 men in four generations of a family of 58 members. The characteristic factor about those cases was the occurrence of a prolonged bleeding time in the presence of a normal number of platelets and a normal coagulation time. Von Willebrand[8,9] first suggested the term "hereditary pseudohemophilia" as applied to this type of bleeding disorder.

Additional reports of hereditary pseudohemophilia have since appeared in the literature. Buckman[4] observed a family in which the disease was transmitted by the males and had occurred in four generations. Minot[7] also reported the transmission of the disease through the male in four generations of a family in which it affected both sexes. Fowler[10] observed two families in which the disease was transmitted by both males and females through several generations. On the other hand, Giffin[11] reported a case in which the disease was transmitted directly by the women through four generations. It has been observed in a Negro by Bailey and McAlpin[12] and appears to be inherited as a dominant Mendelian character residing on the X chromosome. A recent comprehensive review of the literature on pseudohemophilia has been made by Estrin, Sanchez Medal, and Dameshek,[13] who added 11 cases of their own to 62 cases found in the literature in which there was sufficient data to warrant the diagnosis of hereditary pseudohemophilia.

Summary

1. Patients with hereditary pseudohemophilia present the same clinical picture as those having idiopathic thrombocytopenic purpura.

2. The pathogenesis appears to be a defective retraction of the capillaries in response to injury.

3. The hemorrhagic studies show a normal coagulation time, normal platelets, normal clot retraction, and a considerably prolonged bleeding time.

4. There is no successful treatment for this disease, and surgical procedures of an elective nature are contraindicated.

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TANTALUM CRANIOPLASTY IN FRACTURE OF THE SUPRA-ORBITAL RIDGE AND FRONTAL SINUS

Report of a Case

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TANTALUM has proved to be most satisfactory in the closure of cranial defects, as evidenced by the increasing amount of literature on the subject. Tantalum is nontoxic, noncorrosive, inert in tissue, nonabsorbable, and malleable, and is therefore desirable material for alloplastic grafts. This metal has been used with increasing frequency in reparative and restorative surgical procedures about the head and elsewhere in the body. Its value in the reconstruction of traumatic defects involving the periorbital structures and frontal sinuses was demonstrated by Turner[1] in a group of 6 patients, though Conley[2] had previously described its use in a patient with a postoperative defect in this region. Turner noted that small defects in the supra-orbital ridge resulted in disproportionate deformity, and, while the supraciliary arch could be spared in elective surgical procedures, this was not always possible in the extensive comminuted fractures of traumatic lesions. He used tantalum to repair the defect with excellent cosmetic result in the series reported. Tantalum has been used in similar cases at the Cleveland Clinic.

Case Report

A man, aged 24, was admitted to the Cleveland Clinic on September 9, 1947. He had been in an automobile accident on August 31, 1947, when the car in which he was riding struck a tree. He was thrown from the back seat to the front and knocked unconscious for five or ten minutes. The right eye was swollen shut for three or four days, and when it opened the eye was turned downward and outward. He had had moderately severe headaches for several days, which were controlled with aspirin. Roentgenograms had revealed a fracture of the right supra-orbital ridge. The patient was referred to the Cleveland Clinic Hospital for correction of the outward rotation of the eye and the orbital defect.

Past medical history disclosed that at the age of 5 the patient had had a sudden onset of what may have been a right third nerve palsy without other symptoms. He was given corrective glasses, and the eye had improved until it was "almost straight."

General physical examination revealed a well-developed, well-built young man with slight ecchymosis and swelling over the right eyelid. A depression was present in the supra-orbital ridge. The right eye was turned downward and outward, the entire eye being depressed. The blood pressure was 150 systolic, 90 diastolic. The remainder of the general physical examination was negative.

Neurologic examination revealed that, except for a possible slight degree of weakness of the right third nerve, the cranial nerves were intact. The right eye, while being rotated, was obviously depressed as compared to the left but still had a good range of movement. The deep reflexes were normal. No pathologic reflexes were elicited. There were no sensory changes.

On laboratory examination the urinalysis, blood chemistry studies, and blood Wassermann reaction were negative. Blood count demonstrated 5,400,000 red cells, hemoglobin

15.0 Gm. (97 per cent), and 16,000 white blood cells, 58 per cent neutrophils, 2 per cent eosinophils, and 40 per cent lymphocytes.

Roentgenologic examination showed a fracture of the supra-orbital ridge on the right, with an inferior displacement of the fragments. There was a depression of the wall of the frontal sinus and a downward depression of the fragments from the orbital roof. There was no extension of the feature along the other bones of the skull. (fig. 1a and b).

Ophthalmologic examination revealed slight haziness of the optic discs and loss of the inferior field of the right eye.

The patient was admitted to the hospital on September 11, 1947, and a pneumoencephalogram was made to rule out the possibility of a subdural hematoma. Following an encephalogram the patient was prepared for a right frontal operation.

An incision was made within the line of the eyebrow on the right side. A marked depression approximately 1½ cm. deep was present in the right frontal sinus region including the supra-orbital ridge. The bone was fragmented in many pieces, and portions of the mucosa of the frontal sinus were seen extruding between the fragments. The fragments were elevated one by one. It was apparent that the mucosa of the frontal sinus had been lacerated. When the anterior wall of the frontal sinus had been removed the mucosa was stripped from the sinus until no more remained. A gelfoam pad was placed over the nasofrontal duct. Several bone fragments were noted to be turned down into the orbit itself. Because of the multiplicity of

FIG. 1. (a and b) Extensive fracture of right orbital ridge with depression of fragments into orbit; extensive pneumatization of sinuses. (c and d) Postoperative view of tantalum prosthesis in position; note orbital roof projection.

fractures it was impossible to fit the fragments together. A tantalum plate .0007 inch thick was molded to conform to the orbital ridge over the frontal defect. A portion of this plate extended posteriorly at right angles to form a new orbital roof and floor of the frontal sinus. It was tightly fitted and required only a single tantalum screw placed in the right lateral aspect of the incision to hold it in place (fig. 1c and d). A mixture of sulfadiazine and 100,000 units of penicillin was instilled into the sinus cavity. The incision was closed with a double row of buried, inverted, interrupted black silk sutures. The right eye, which had previously deviated downward and outward, had again resumed its normal position in the head.

The patient was given penicillin, sulfadiazine, and neosynephrine nasal sprays for ten days and had an uneventful postoperative course. He was discharged from the hospital on September 25, 1947, with instructions to continue the nasal spray.

On October 31, 1947, he noted a slight swelling over the plated area. A small amount of fluid was aspirated from beneath the skin, cultures and smears of which were negative. He was given penicillin prophylactically and was instructed to use a neosynephrine spray. The patient improved rapidly, and the swelling subsided within a day or two. When last seen seven months later he had resumed his full normal activity without further complications (fig. 2).

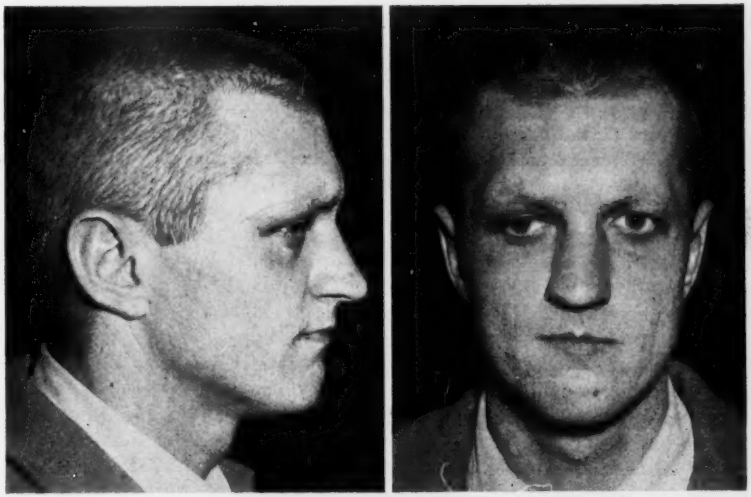

FIG. 2. Postoperative appearance of right orbital reconstruction. Lateral and frontal views.

Discussion

In this case the external violence fractured the heavy supraciliary ridge downward into the orbit and the anterior wall of the large frontal sinus into the sinus cavity. Since the tissue overlying the defect appeared in good condition the operation was performed early, though Gardner[3] believes that the presence of a tantalum implant does not impair the healing of an infected wound. Pneumoencephalography was carried out to reject the possibility of other intracranial disease, a procedure done by Turner before each cranioplasty.

It is important to reproduce the convexity of the vertical or frontal limb as well as the convexity of the orbital portion which extended backward at approximately right angles. This approximation was most accurately obtained by comparison with the anterior-posterior and lateral roentgenographic views of the head. Because of the malleability and the lightness of the tantalum sheet used, no difficulty was encountered in obtaining the necessary tight fit. After the plate was molded it was locked in place with only one laterally placed tantalum screw. Bearing in mind the experience of Woodhall and Cramer[4] with extradural pneumatocele in tantalum cranioplasty in which a fistulous tract extended from the frontal sinus into a fibrous sac enveloping the tantalum plate, no perforations were made in the plate. The small collection of fluid which appeared one month later might have been avoided had perforation been present, though it seemed to be the lesser of two evils.

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THE USE OF TANTALUM TUBES IN FRONTAL SINUS SURGERY

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OPERATIONS on the frontal sinus at times produce unsatisfactory results. Analysis of the causes of failure is helpful, but improved technics are still needed.

Among the causes of failure of the radical frontal operation are:

(1) Incomplete removal of the floor of the frontal sinus which may result in a partitioning and formation of pockets in the remaining portion of the sinus cavity.

(2) Incomplete ethmoidectomy leaving the orbital ethmoid cell extension undisturbed.

(3) Removal of the frontal process of the superior maxilla, allowing collapse of the nasofrontal passage from pressure of the orbital soft tissues.

Even when careful technic is observed and complete removal of the floor of the frontal sinus and the ethmoid cells is accomplished as in the Lynch operation for ethmoid and frontal chronic sinusitis, 20 per cent of the operations result in failure because of adhesions and closure of the nasofrontal duct.

Several precautions should be taken to prevent closure of the nasofrontal passage. An opening is usually made through the nose into the frontal sinus. It should be large enough to ensure subsequent drainage of the sinus when healing has occurred. At the time of operation the communication appears unnecessarily large, but the processes of healing render it incredibly small.

The tendency to closure may be prevented by leaving the frontal process of the maxilla undisturbed, thus preventing the orbital periosteum from moving mesially. Patency of the opening is best assured by the use of tantalum tubes. These tubes are inert in the tissues and do not interfere with epithelization. The tube is placed in the newly created nasofrontal communication with the upper end in the frontal sinus and the lower end protruding from the nostril. The presence of the tube prevents granulation tissue from covering surfaces denuded of epithelium and from bridging and obstructing the nasofrontal passage. If contact of the granulation tissue on opposite sides can be prevented, complete epithelization of the surfaces will occur. Tantalum tubes are light in weight, cause no discomfort, and provide free exit for secretions from the frontal sinus. Since there is no reaction in tissue surrounding the tantalum tube, such as edema of the mucous membrane and formation of granulation tissue, secretions do not become "sealed off" but continue to drain through and around the tube.

Welded tantalum tubes* 5 mm., 7½ mm., and 10 mm. in diameter are used to accommodate the space between the frontal process of the maxilla,

**The tantalum tubes were supplied by the Ethicon Division of Johnson & Johnson.*

the interfrontal septum, and the spine of the frontal bone. The 10 mm. tube is used when the frontal spine has been removed at a previous operation or when its removal has been necessary because of infected bone or to expose the anterior ethmoid region. Occasionally, the frontal spine has disappeared due to erosion caused by pressure of a mucocele.

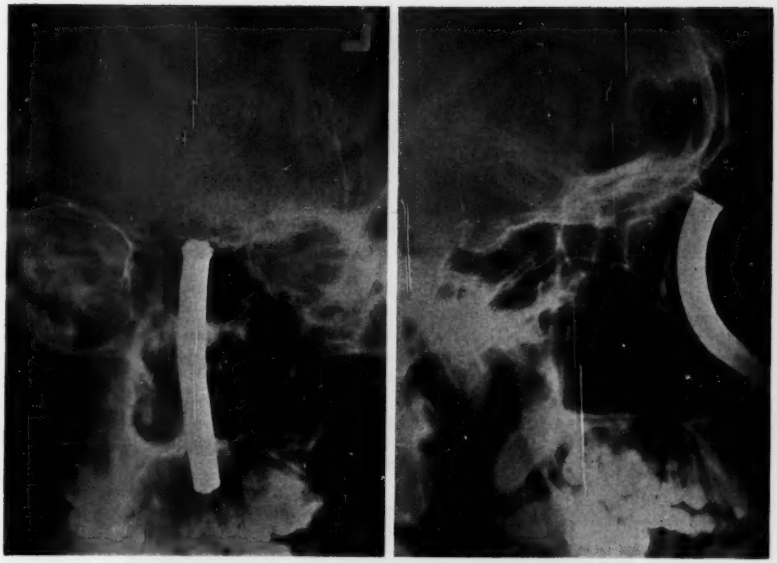
The tantalum is fixed in position by a No. 28 tantalum wire, which is introduced through a small hole 1 mm. from the end of the tube. The two ends of the wire are drawn out through the skin incision and anchored to the forehead by adhesive tape or by a wire suture.

Following operation the tube is cleaned by means of inserting a small curved metal suction tube or a small sterile rubber catheter connected to a suction machine. The tantalum tube remains in position from four to six weeks, depending upon conditions of the nasofrontal passage. At the end of this time epithelization is completed and a well healed nasofrontal communication has resulted.

In 1945 Goodale[1] reported his experience with sheets of tantalum foil to prevent the surface of the orbital periosteum from coming into contact with

a.

FIG. 1. Case 1. (a) Preoperative roentgenogram reveals bilateral large orbital ethmoid extensions with cloudiness of frontal sinuses.



b.

FIG. 1. Case 1. (b) Postoperative anteroposterior and lateral films of the frontal and ethmoid sinuses reveal tantalum tube anchored with tantalum wire in nasofrontal passage.

the medial wall of the nasofrontal passage. He found that when the tantalum foil was used the nasofrontal passage remained open.

Case Reports

Case 1. A man, aged 42, entered the Cleveland Clinic on July 25, 1947, with the complaint of "frontal sinus trouble" present since 1922. He had undergone three operations upon the frontal sinus by the external approach, the first in 1928 and the last in 1940. He had suffered from intermittent frontal headaches and recurrent swelling above the left eye. In 1946 a fistula had developed in the mid-supraorbital region.

Preoperative roentgenograms revealed bilateral, large, orbital, ethmoid cells with cloudiness of both frontal sinuses.

A radical left external frontal operation was performed, removing the entire floor of this sinus, all of the lining membrane, a portion of the anterior wall which showed dehiscence and evidence of localized osteomyelitis, and all of the left ethmoid, including the large orbital ethmoid cells, which contained pus and thickened mucous membrane. The right frontal sinus was entered through the interfrontal septum, and only mild thickening of the lining mucous membrane was encountered. No further procedure was carried out on the right frontal sinus. A curved tantalum tube 7½ mm. in diameter was placed in the left nasal cavity through the newly created nasofrontal opening, with the upper end resting on the floor of the frontal sinus. The lower end of the tube protruded 4 mm. from the external naris. A tantalum wire was inserted through a small hole near the upper margin of the tube, was brought out through the external incision, and was anchored to the skin of the forehead with adhesive tape. The wire was taut to prevent the tantalum tube from sliding out of position.

The tube was removed during the fifth postoperative week. The external fistula has remained healed, the new frontal opening is large and patent, and the patient is entirely free of symptoms (fig. 1).

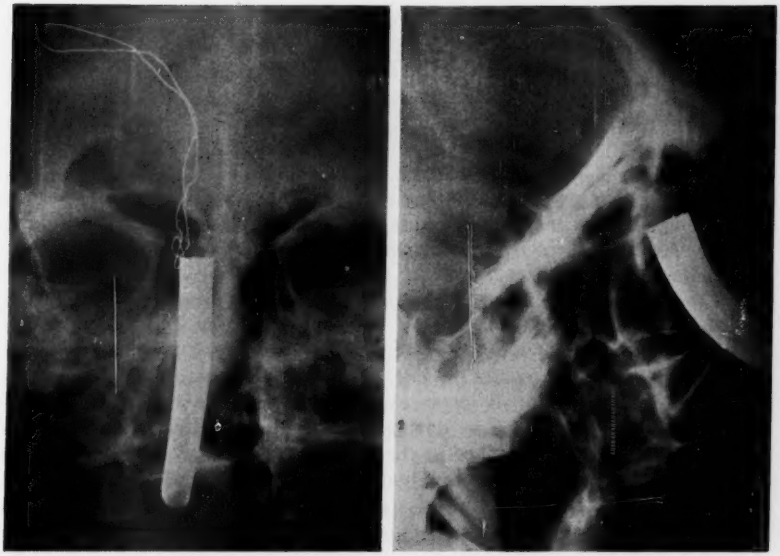
Case 2. A man, aged 35, was admitted to the Cleveland Clinic on February 1, 1945, complaining of a discharging fistula over the left eye and diplopia following a left external frontal operation in February, 1943. Nine years previously he had had severe pain over the left eye, lasting for one month. This was followed by a bilateral purulent nasal discharge which had continued until present admission. He had received intense sulfa and penicillin therapy without significant improvement.

Preoperative roentgenograms revealed cloudiness of all the paranasal sinuses. On February 5, 1945, a bilateral intranasal antrotomy was carried out to eliminate the maxillary sinus infection. This was followed by a left external frontal operation (Lynch procedure). The orbital ethmoid extensions were removed and found to be filled with thickened mucous membrane and pus. The supraorbital fistula extended through a dehiscence in the floor of the frontal sinus, which, with all of the lining membrane, was completely removed. A tantalum tube was placed in the nose, with the upper end resting in the frontal sinus according to the method described in case 1. The patient made a prompt and complete recovery and has remained symptom-free. The nasofrontal passage now appears well epithelized and is free of granulations and surrounding edema.

Case 3. A man, aged 32, was admitted to the Cleveland Clinic with the complaint of intermittent swelling above the right eye of two years' duration. Trauma to the right frontal region in 1934 had been followed by frontal headaches, purulent anterior and posterior

a.

FIG. 2. Case 3. (a) Preoperative roentgenogram reveals cloudiness of right frontal sinus with absence of part of bony floor and anterior wall. Right ethmoid area is clouded by mucocele which protrudes into nasal cavity. Arrow points to nasal border of mucocele.



b.

FIG. 2. Case 3. (b) Postoperative roentgenograms show tantalum tube anchored *in situ.*

nasal discharge, and nasal obstruction on the right side. One year after the injury the patient had had a bilateral external frontal operation. In 1938 bilateral Caldwell-Luc operations and a second right external frontal operation had been performed.

Physical examination on admission revealed diplopia and edema of the right upper eyelid with closure of the palpebral fissure. Roentgenologic examination demonstrated a mucocele of the right frontal sinus with partial absence of the bony floor and anterior wall. At operation the usual Lynch procedure was carried out, with removal of the mucocele and mucous membrane lining the sinus cavity. A large tantalum tube was placed in the frontal sinus through the right side of the nose, as described in case 1, and anchored in the usual manner. The patient was discharged from the hospital with the tube in place. Upon follow-up examination eight weeks later the tube was removed and all swelling had disappeared. The patient has remained free of symptoms (fig. 2).

Summary

In spite of careful technic in surgery of the frontal sinus, postoperative closure of the nasofrontal passage may give rise to failure. A method involving the use of tantalum tubes will maintain a permanent communication between the frontal sinus and the nasal cavity and thus decrease the number of unfavorable results from this type of surgical procedure.

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THE TREATMENT OF CONGESTIVE HEART FAILURE

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CONGESTIVE myocardial failure is one of the most important problems in clinical medicine. It may result from any form of heart disease. Among the less common causes are cardiac conditions secondary to hyperthyroidism, myxedema, beriberi, severe anemia, and arteriovenous aneurysm. In this group the principal measures of treatment are directed toward correcting the underlying disorder. The majority of cases, however, are consequent to the more common types of heart disease. In these, treatment is not determined by the etiology of the condition but consists of well established procedures which are applicable in all instances. Intensive research on the pathologic physiology of congestive failure during the past few years has resulted in the adoption of important changes in therapy which in turn have considerably improved the outlook of the individual patient.

Rest. A period of rest in bed is an essential part of the management of every case of congestive heart failure. Levine[1] has pointed out that the sudden enforcement of the conventional type of bed rest may have certain harmful effects. These are to be regarded as complications against which suitable precautions must be taken, and the possibility of their occurrence does not detract from the importance of rest itself. In patients with congestive failure, the recumbent position, through the effect of gravity, brings about a shift of edema fluid from the lower to the upper portions of the body. This often results in prompt diminution in the edema of the lower extremities, but, unless proper treatment has been instituted, edema may simultaneously appear or increase over the back, and the evidence of pulmonary congestion may become more apparent. Hydrothorax may develop for the first time. The recumbent position also facilitates the return flow of blood to the heart and, by favoring absorption of edema fluid into the blood stream, results in an increase in the circulating blood volume.[2] The load upon the heart is therefore augmented, and unless corrective steps are taken a decided increase in the degree of failure may result. These undesirable effects can be avoided by prompt and vigorous treatment with digitalis and diuretic drugs and, in patients with severe decompensation and considerable edema, by the use of shock blocks or kitchen chairs under the head of the bed. The latter measures were proposed by Levine, and their use readily convinces one of their value. Occasionally it may be advisable to allow the patient to spend the first few days in a comfortable chair with the feet dependent, but just as soon as improvement has been initiated, he should be transferred to bed.

It is our opinion that in every case of congestive heart failure of more than slight degree, bed rest should be continued for at least six weeks. If there is difficulty in the use of the bed pan and the patient is improving satisfactorily,

the program may be modified after ten days or so by allowing the use of a commode or a daily trip to the bathroom, but no other exceptions should be made. After completion of the period in bed, gradually increasing activity is permitted, but precautions must be taken to avoid dyspnea and fatigue, and there must be careful observation for a gain in weight and any return of edema.

Digitalis. Digitalis remains the most valuable drug in the treatment of congestive heart failure. Every patient in failure should be completely digitalized, and the state of digitalization should then be maintained permanently. Digitalis leaf may be administered in pills, tablets, or capsules whose potency has been adjusted to conform to the international standard, or one may employ one of the purified glycosides, such as digitoxin. Digitoxin has the advantages of causing less gastric irritation than do preparations of digitalis leaf and of being susceptible to chemical assay. The former feature makes it possible to digitalize patients rapidly without causing nausea and vomiting. It has become our practice, in individuals who have not been taking digitalis previously, to begin treatment by the administration of 0.8 mg. of digitoxin, followed in six hours by a dose of 0.4 mg. This constitutes the average amount of the drug necessary to digitalize a patient.

When auricular fibrillation is present the ventricular rate furnishes the best guide as to the completeness of digitalization, provided the patient does not have hyperthyroidism. If, in the absence of thyrotoxicosis, the ventricular rate is still above 70 beats per minute on the day after beginning treatment, digitoxin is prescribed in doses of 0.2 mg. twice daily or digitalis in doses of 0.1 Gm. two or three times daily. This schedule is continued until the desired control of the ventricular rate has been accomplished or the first evidence of digitalis overdosage appears. The dose is then reduced to 0.2 mg. of digitoxin or 0.1 Gm. of digitalis on five to seven days of each week, and this maintenance amount is continued indefinitely.

When the heart rhythm is normal, the ventricular rate cannot be used as a guide to the degree of digitalization. Digitoxin, therefore, is given according to the initial schedule outlined above, and on the second day of treatment the patient is placed upon a maintenance dose of either digitoxin or digitalis. In large individuals a somewhat greater amount, such as digitoxin 0.2 mg. twice daily or digitalis 0.1 Gm. two or three times a day, may be continued for a day or two. Overdosage with digitoxin causes nausea, vomiting, and other manifestations of intoxication, just as do excessive amounts of digitalis. As in the case of digitalis, these symptoms probably are the result of a direct toxic effect of the drug on the heart.

It may be objected by some that rapid digitalization as accomplished by administering digitoxin according to the outline given is not necessary in the majority of cases of congestive failure. Most patients who have decompensation are seen before the condition has reached a far advanced stage, and in the past they have responded satisfactorily when the process of digitalization has been spread over a period of several days. However, if the maximum therapeutic effect of the drug is obtained more rapidly by the use of digitoxin, the patient begins to improve more promptly and becomes comfortable earlier.

Furthermore, in individuals who have extensive edema, rapid digitalization helps greatly in preventing the possible untoward effects of suddenly instituting a program of strict rest in bed.

Except for the uncommon arrhythmia ventricular paroxysmal tachycardia, there are no contraindications for the use of digitalis when congestive heart failure is present. In patients who have partial heart block of either first or second degree, however, and in those who have frequent ventricular premature beats, digitalization should be accomplished gradually and the electrocardiogram should be checked daily.

The oral route of administration is the one of choice for digitalis and digitoxin and can be employed in all but an occasional patient. At times a condition is present which causes vomiting or prevents the taking of drugs by mouth, and intramuscular administration must be resorted to. Several preparations are available for use in this manner. For those which contain 1 cat unit of digitalis in 2 cc. of solution, an initial dose of 10 cc. can be given and can be followed in four to six hours by a second injection of the same amount. The process of digitalization is then completed by administering 2 cc. two or three times a day until the full therapeutic effect of the drug is obtained or evidence of overdosage appears. Whenever digitalis is given by any route, it is desirable, of course, to avoid the production of toxic symptoms.

The absorption of digitalis from muscular tissue is no more rapid than is absorption from the gastrointestinal tract. In true cardiac emergencies, therefore, where a delay of even a few hours in securing the effect of the drug might mean the difference between a successful and a fatal outcome, intravenous administration of a digitalis preparation is indicated. Cases of this kind are uncommon. The preparations mentioned for intramuscular use may also be given by intravenous injection, and the dosage is the same by either route. If one prefers, ouabain, digitoxin, or cedilanid may be employed. When ouabain is used, the initial dose is usually 0.5 mg., and this is followed by additional injections of 0.1 mg. to 0.25 mg. every four to six hours until a total of not more than 1.0 mg. has been administered. For digitoxin, intravenous dosage is the same as for oral administration, namely 0.8 mg. (4 cc.) followed in four to six hours by 0.4 mg. If cedilanid is employed, the usual schedule is 0.8 mg. (4 cc.) followed in four to six hours by a second injection of 0.4 mg. to 0.8 mg. After one of these schedules has been finished, the process of digitalization is completed by oral administration of digitalis or digitoxin or, if this is not possible, by intramuscular injection of a suitable preparation in the manner already outlined. It is, of course, essential to be certain that patients to whom ouabain or digitalis preparations are to be given intravenously have not received digitalis during the preceding two weeks.

Diet. The most significant change in the treatment of congestive heart failure in recent years has been the introduction and general use of diets of restricted sodium content. Formerly it was customary to limit the fluid intake of patients suffering from cardiac decompensation and pay little attention to the amount of salt in the food. This has been changed entirely as a result of repeated demonstration that sodium retention, probably on a renal basis,

is a cardinal feature of congestive failure and the most important factor responsible for water retention and the development of edema. There is quite uniform agreement today that the diet of patients with myocardial failure should contain less than 2 Gm. of sodium chloride per twenty-four hours. Restriction of the fluid intake is unnecessary and may even be harmful. The most satisfactory results are obtained when the patient takes between 2 and 3 liters of water daily, and whenever an individual is unable to drink this much, it appears advisable to administer sufficient 5 per cent glucose solution in distilled water by intravenous drip to bring the total fluid intake up to the desired level.

The preparation of a low sodium diet can be accomplished in the home without great difficulty if specific instructions are given. No salt is used in cooking, all salted foods are eliminated, and only salt-free bread and unsalted or washed butter are allowed. Canned foods to which salt has been added during processing and all foods prepared with baking soda or baking powder are prohibited. Medicines and proprietary preparations which contain sodium must not be used. A sodium-free salt substitute may be taken if desired.

Diuretics. Although many individuals who have congestive failure will recover satisfactorily when treated by means of rest, digitalis, and a low sodium diet without other measures, the additional use of diuretic drugs hastens improvement and more promptly restores the patient to a state of comfort. Their administration, therefore, is indicated in every case. The most effective preparations for routine use are the organic mercurial compounds, and of the members of this group mercuhydrin is the one most extensively employed at present. This preparation is equal to the other members in diuretic action and is less toxic. It can be given by intravenous or intramuscular injection, but the intramuscular route is preferred because of its greater safety. It was customary formerly to administer the drug at intervals of three to five days, but Gold and his associates[3] are of the opinion that such intervals between injections serve no useful purpose. They recently recommended daily administration until no further diuresis results and the patient's weight remains stationary. No undesirable effects were observed from this procedure in a large series of cases, and they are convinced, therefore, that daily injections may be given safely to all patients in whom the output of urine is within normal limits. An initial dose of 0.5 cc. is advised, and this is increased to 1.0 cc. and then to 2.0 cc. whenever the individual fails to lose 3 pounds in twenty-four hours. After all edema has disappeared and the daily injection fails to cause further diuresis and loss in weight, the interval between doses is lengthened gradually, and in favorable cases the drug is eventually discontinued.

When mercurial diuretics are employed, no advantage results from the simultaneous administration of ammonium chloride by mouth. The latter preparation is helpful at times, however, in doses of 4 to 8 Gm. daily, in preventing a return of edema after the patient has become ambulatory. In other patients, synthetic theophylline in doses of 0.1 Gm. three times a day is effective when given for the same purpose.

Morphine and Sedatives. Persons suffering from congestive failure of moderate or severe degree frequently have gone for many nights with little restful sleep, and this contributes considerably to their exhaustion and apprehension. In all such cases, morphine should be administered either when the patient is first seen or in the evening of that day. A night of comfortable sleep often produces a remarkable improvement in general condition and morale. In severe failure, the use of morphine may be necessary on the following two or three nights, but after this milder preparations such as one of the barbiturates usually suffice. Mental as well as physical rest is important, and in restless, worried, or emotionally tense individuals a mild sedative, such as a small dose of phenobarbital, is advisable two or three times a day during the early part of treatment.

Venesection and Thoracentesis. In myocardial failure involving the right as well as the left ventricle, the peripheral venous pressure is increased approximately in proportion to the degree of decompensation. When failure is severe, the jugular veins often are engorged to the angle of the jaw even with the patient sitting upright. Venesection should be performed in such instances with the removal of 500 cc. or 600 cc. of blood, and the same procedure should be carried out in less advanced cases whenever digitalis therapy and the other measures mentioned previously fail to produce a satisfactory response. Venesection directly reduces venous congestion and diminishes the degree of dilatation of the heart. In favorable cases, the venous pressure remains low after having been reduced by the removal of blood, but in unfavorable situations peripheral venous congestion promptly returns.

Advanced congestive failure often is attended by the accumulation of large amounts of fluid in one or both sides of the thorax. The resultant compression of the lungs further reduces the already diminished vital capacity and by so doing increases the degree of dyspnea. Whenever pleural effusion of moderate or greater degree is present, therefore, the fluid should be removed as completely as possible by prompt thoracentesis.

Estimation of Clinical Progress. There are a number of guides which can be used in estimating the patient's response to treatment. Of these, the most important are relief from such symptoms as dyspnea, cough and malaise, the occurrence of diuresis, disappearance of clinical edema, clearing of the evidence of passive congestion in the lungs and liver, improvement in the vital capacity of the lungs, return of the peripheral venous pressure to normal, control of the ventricular rate when auricular fibrillation is present, and the course of the weight curve. It must be remembered that a considerably increased volume of extracellular fluid may still remain in the body after all apparent edema has disappeared and the patient has become comfortable. Convalescence cannot be considered satisfactory until this subclinical edema also has been eliminated. This is one of the reasons why Gold places great emphasis on the patient's weight curve and is the main consideration for recommending the use of mercurial diuretics at whatever intervals and for whatever length of time are necessary to maintain the minimal weight and a relatively constant output of urine.

In conclusion, although the majority of patients who have had congestive heart failure eventually die of a recurrence of the condition or of some other complication of the underlying heart disease, the treatment of myocardial failure should not be a matter of pessimism. Management according to the principles which have been outlined will restore many patients to a useful and self-supporting state for long periods of time and will contribute greatly to the comfort of the less fortunate ones who cannot be helped beyond a life of semi-invalidism.

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VENOUS MESENTERIC THROMBOSIS

Report of a Case with Recovery

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RECOVERY from venous mesenteric thrombosis is rare. D'Abreu and Humble[1] state that the prognosis in thrombosis of the mesenteric arteries is more favorable than in cases involving the veins.

The case reported here demonstrates the following unusual features: (1) recovery from venous mesenteric thrombosis, (2) construction of an artificial external anastomosis of the small bowel, and (3) use of a spur clamp on the small intestine with subsequent closure as in a Mikulicz resection.

Case Report

An obese white man, aged 60, was admitted to the Cleveland Clinic Hospital on July 29, 1947, with sharp pain in the right upper quadrant of the abdomen. Four months prior to admission he had experienced intermittent, dull, aching pain in the lower quadrant of the abdomen, and one month before admission he had entered another hospital because of sudden, severe, sharp, intermittent, midabdominal pain. No definite diagnosis was established, and the patient gradually improved and was discharged two weeks later. Ten days prior to admission he had had severe diarrhea without blood or mucus which lasted three days. The bowel habit was normal at the time of admission.

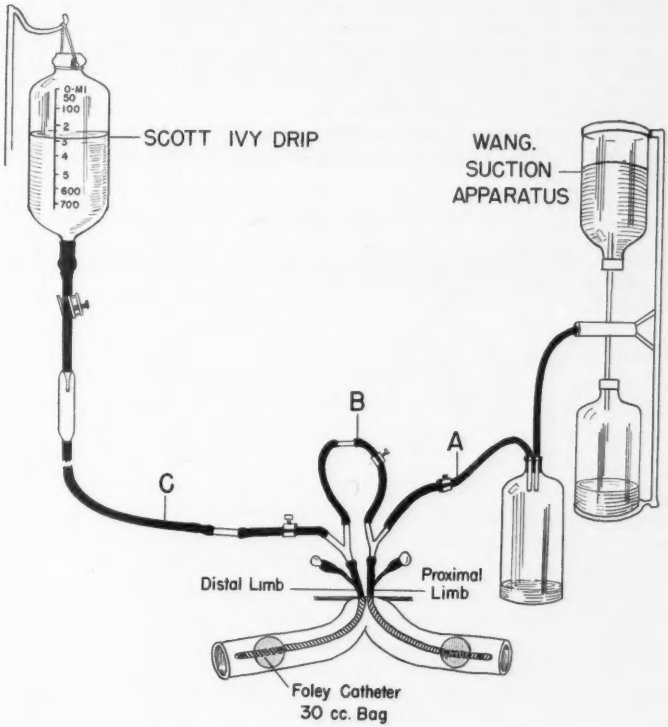
On physical examination the temperature was 101.3 F., pulse 110, respirations 28, and blood pressure 120 systolic, 80 diastolic. The upper portion of the abdomen was hyperresonant and the lower portion dull. A large mass was palpable in the right lower quadrant. Peristalsis was present, but sounds were infrequent. Laboratory studies did not reveal any abnormalities except a white blood count of 9300 with 87 per cent polymorphonuclears.

The patient was given supportive therapy, and a Harris tube was introduced but failed to pass through the pylorus. A definite diagnosis was not established, although a perforated appendix with abscess formation was suspected.

Operation was performed on July 31, 1947, under spinal anesthesia induced by 10 mg. pontocaine and 100 mg. procaine hydrochloride, supplemented by endotracheal ether. A McBurney incision was made, releasing a large quantity of thin bloody peritoneal fluid. Exploration disclosed a gangrenous segment of the distal ileum. The wound was closed, and a midline incision was made. Thirty-two inches of lower ileum containing the gangrenous segment was resected, and an isoperistaltic anastomosis was constructed. The left side of the abdomen was then explored, and a loop of the distal jejunum was found adherent to the parietal peritoneum. This was freed, exposing a three-inch loop of completely necrotic jejunum. Because of the condition of the patient and the uncertainty of the viability of the intestine in this area, the loop of necrotic jejunum was exteriorized and the necrotic bowel and mesentery trimmed away. Catheters were tied in the proximal and distal limbs of the exteriorized bowel. The wound was closed with figure of eight alloy steel sutures. During the operative procedure 1000 cc. of whole blood was administered intravenously.

The abnormalities revealed at operation and by examination of the pathologic specimen were (1) recent and extensive venous mesenteric thrombosis of two feet of the terminal ileum with hemorrhagic infarction, (2) old established venous mesenteric thrombosis with gangrene of three inches of the lower jejunum, and (3) hemorrhagic ascites (1000 cc.).

Postoperative Course. The patient was given heparin by continuous intravenous drip through a plastic tube. The coagulation time fluctuated widely in spite of repeated attempts at stabilization. Dicumarol was administered simultaneously. The prothrombin time was prolonged satisfactorily in forty-eight hours, and the heparin was then discontinued. The jejunostomy was functioning satisfactorily forty-eight hours after operation. However, it was difficult to maintain chemical balance because of electrolyte loss, and one week after operation an artificial external anastomosis was made (figure) to maintain electrolyte balance. The



Legend

Diagram of apparatus. A No. 24 Foley catheter (30-cc.-bag capacity) was inserted into each limb of the double-barrelled jejunostomy and a Y-glass tube placed in each catheter. Tube A was attached to a Wangensteen suction apparatus for use as needed. Tube B was attached through a glass adapter to the distal limb. Peristalsis forced the intestinal content through the anastomosis freely as long as the oral intake was restricted to clear liquids. Tube C was attached to a bottle (Murphy drip) for intermittent Scott-Ivy feedings. A series of stopcocks facilitated the use of the anastomosis.

The authors express their thanks to Miss Beatrice Boyle for her drawing of the apparatus.

patient developed a progressive icterus which reached a peak of 70 three weeks after operation. During the administration of dicumarol the prothrombin time varied considerably. In a little more than three weeks after operation anticoagulant therapy was discontinued.

The limbs of the jejunostomy were side by side, and approximately four weeks after operation a spur clamp was applied to the septum between the limbs. The clamp fell away five days later. The patient was having bowel movements per rectum, but leakage at the jejunostomy was so great that nutrition could not be maintained without supplemental intravenous therapy. (Throughout the patient's hospital stay penicillin, intravenous fluids, amino acids, transfusions, vitamins, and digitalis were given as indicated.) Approximately seven weeks after the initial operation the jejunostomy was closed under spinal anesthesia. The bowel was closed with inverting continuous chromic sutures and the abdomen with through and through steel sutures. After closure there was slight leakage of intestinal content at the suture line, and sump drainage was instituted. Drainage ceased on October 10, 1947, and the patient was discharged on the following day.

He returned on February 4, 1948, at which time he was in good general health but had a small incisional hernia. The patient was given permission to return to his occupation as a watchman

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MEASURES OF RENAL TUBULAR FUNCTION

Comparison of the Addis and Pituitrin Concentration Tests

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ESTIMATION of the ability of renal tubules to concentrate urine is of as great value as any single test in establishing diagnosis and prognosis of renal disease. Further, it can be performed accurately and simply, at no cost to the patient and with minimal effort to the examiner. With this to recommend it, the principle should be widely applied.

In the measurement of concentrating ability the aim should be to determine maximal or "ceiling" specific gravity. Possibly because many of the methods suggested do not furnish such values they are infrequently used. General acceptance and usage of a single, practical method of testing for maximal specific gravity might contribute toward more uniformity in the understanding of the various types of renal disease.

The method described by Lashmet and Newburgh[1] probably most nearly measures maximal concentrating ability. They proposed that the oral intake of solids be controlled for seven days before and three days during water deprivation. However, the duration and rigorous restrictions of the test make routine application unlikely.

Brunn, 1921;[2] Sodeman and Engelhardt, 1941-1943;[3,4] and more recently Horne and Morris, 1947,[5] have utilized the antidiuretic principle of posterior pituitary extract as a means of estimating the ability of the kidneys to concentrate urine. After subcutaneous injection of 10 units of surgical pituitrin, they observed a sharp decrease of urinary output and an increase of specific gravity which attained its maximum degree at two hours and persisted for three to four hours. The specific gravities of the specimens collected from normal persons ranged from 1.022 to 1.036 (Sodeman and Engelhardt)[4] and 1.020 to 1.029 (Horne and Morris).[5] These authors have suggested that the convenience of the test and the results obtained might justify substituting it for tests that depend upon fluid deprivation.

Taylor, Peirce and Page[6] compared the results obtained by the method described by Sodeman and Engelhardt to those recorded following a period of 24 hours fluid deprivation (Addis).[7]

They found that injection of pituitary extract into normal subjects yielded urinary specific gravities of 1.015 to 1.033, a spread only slightly greater than that reported by proponents of the test. On the other hand, the Addis test, as has been previously shown by its author and by Alving and Van Slyke[8] produced specimens whose specific gravities among normal persons always exceeded 1.026.

Comment

Since the antidiuretic action of posterior pituitary extract is maximal two hours after injection, the pituitary tests can be performed in a short period without preparation. However, this apparent advantage may be offset by the inconveniences imposed by volumes of urine formed during the two hour period of oliguria induced by pituitrin. Many patients will form but 5 to 15 cc. of urine under such conditions, a volume which prevents measurement of gravity by an urinometer. In such instances a pyknometer and analytical balance must be used. Further, scanty amounts of urine may make quantitative estimation of proteinuria difficult. Without this information an accurate specific gravity determination is impossible, for 1 per cent proteinuria can elevate the specific gravity 0.003.

In addition to these mechanical disadvantages of the pituitary test, patients must spend at least two hours waiting in a laboratory or office, while the Addis test may be performed by almost anyone during normal activity. Simple instructions are rarely misinterpreted. The test requires only fluid deprivation for twenty-four hours and collection of the urine formed during the second

Instructions for Addis Test

1. No fluid or frozen foods of any kind from 8 a.m. until 8:00 a.m. of following day.
2. Dry diet is permissible.
3. At 8:00 p.m. empty bladder and discard urine.
4. Save all urine passed from 8:00 p.m. until 8:00 a.m.
   Keep in jar in icebox or cool place.
5. Bring specimen to desk from which list of instructions was received.

twelve hours of this period. Normal subjects produce specimens with specific gravities which range from 1.026 to 1.032. Further, the specimen is always large enough so that its gravity can be measured quickly with a urinometer and its protein content determined. If indicated, an Addis count[7] of the sediment can be made.

Although this test of concentrating ability seems to be the most satisfactory for routine use, there are cases in which it has limitations. Patients with diabetes insipidus or edema, regardless of origin, may produce dilute urine regardless of the degree of dehydration and thus vitiate the Addis test as a measure of renal efficiency. If a test of renal function is desired among such patients, the urea clearance serves admirably.

Interpretation of Urinary Specific Gravity Measurements

Values of urinary specific gravity which do not approximate the "ceiling" or maximal value for the individual patient can have little meaning save in a

very broad sense. In the early or moderately advanced stages of most renal disease and especially in chronic glomerulonephritis, pyelonephritis, and essential or malignant hypertensive vascular disease, it is commonplace to have isolated urine specimens with specific gravities of 1.020 or above. This is possibly the result of relative dehydration occurring by chance. In those tests which depend upon the effects of pituitary extract on the kidneys or brief periods of fluid restriction there is no way of separating this type of patient from the normal as far as tubular function is concerned. Patients to whom this might occur are those in whom accurate estimation of urinary concentrating ability is of most value. If the disease is far enough advanced to permit formation of urine with specific gravity constantly below 1.020, even the crudest tests or the history of frequency and nocturia are adequate evidences of extensive tubular deterioration.

The interpretation of tests of concentrating ability must naturally be made in relation to other observations in the patient. For instance, retinal arteriolar sclerosis, arterial hypertension and cardiac hypertrophy may be present in similar degrees in a patient with chronic glomerulonephritis and one with essential hypertension both of whom concentrate urine to 1.021. The former, however, will have proteinuria usually in excess of 3-4 Gm. per twenty-four hours and the urea clearance will be depressed to a considerable degree, while these two findings may be within normal limits in the patient with essential hypertension. In those patients with pyelonephritis, acute glomerulonephritis and the malignant phase of hypertension, the degree of pyuria, hematuria or cylindruria may be the determining factor in establishing differential diagnosis.

Since renal and vascular diseases vary in their rate of progression from patient to patient, an isolated test of concentrating power is of limited value for purposes of prognostication. The rate of deterioration can be known only by serial observations made at intervals spaced according to the nature of the disease involved. In the cases of patients with early essential hypertension this information is of more than academic interest. At present, probably the best indication for lumbodorsal sympathectomy as treatment for these patients is evidence of advancing arteriolar disease (Page, Corcoran, and Taylor).[9] Corcoran and Page[10] have indicated that one of the earliest signs of such change is diminishing tubular function which can well be demonstrated by measuring the ability of kidneys to concentrate urine.

Summary

1. Estimation of "ceiling" or maximal ability of kidneys to concentrate urine is a reliable test for renal functions.

2. The Addis test is inexpensive and convenient. It approximates "ceiling" specific gravity and sharply segregates abnormal and normal values.

3. Single measurements of maximal concentrating ability are of diagnostic value but for prognosis in chronic progressive disease of the kidneys serial estimates at six to twelve month intervals are necessary.

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TREATMENT OF GASTRIC ULCER

The Difficulty of Differentiating Peptic Ulcer from Carcinoma of the Stomach

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Division of Surgery

PRIOR to Dragstedt's popularization of vagotomy, gastric ulcer usually was treated by gastric resection. The results of gastric resection for gastric ulcer were good, and the operation afforded maximum protection against the presence or development of carcinoma.

Recently the good results that have been obtained by vagotomy in the treatment of duodenal ulcer and marginal ulcer have led to its use also in cases of gastric ulcer. Our experience with vagotomy in a small series of selected cases of gastric ulcer has been disappointing and has shown the danger inherent in assuming that any ulcerating lesion of the stomach is benign. Two cases are presented in tabular form to emphasize the difficulty of distinguishing between benign and malignant ulcers of the stomach and to point out the importance of removing the ulcer whenever it is possible to do so.

In one case a shallow ulcer, 0.5 cm. in diameter, appeared to be healing and was not demonstrable either by roentgenogram or by gastroscopy. Both the surgeon and the pathologist considered it to be benign until permanent pathologic sections proved it malignant. One side of this lesion was a benign ulcer showing no evidence of carcinoma, and it was from this area that the biopsy specimen was taken. However, permanent sections showed the other side to be an infiltrating carcinoma. In a second case a huge deep ulcer measuring 6 cm. by 4.5 cm., which the roentgenologist and the surgeon had assumed to be carcinoma, proved to be benign.

The Nature of Gastric Ulcer

Gastric ulcer and duodenal ulcer have different consequences and require different treatments. The treatment of gastric ulcer is primarily surgical, involving excision of a potential cancer, whereas the treatment of duodenal ulcer is essentially medical and requires operation only when the symptoms are rendered intractable by complications. Moreover, we must not assume that because vagus resection with gastroenterostomy or pyloroplasty is the preferred surgical treatment for duodenal ulcer that the same treatment is equally applicable to gastric ulcer.

In patients with gastric ulcer the acidity of the gastric juice tends to be normal or even low. After a gastroenterostomy for gastric ulcer there is little or no tendency for ulceration to recur in the jejunum. Gastric ulcer is less common than duodenal ulcer, attacks people of an older age group, and affects women more often than does duodenal ulcer.

The results of gastric resection for gastric ulcer are excellent. The mortality rate is low because there is no problem of closing the duodenal stump. Marginal ulcer is exceedingly rare. The resections need not be so radical as in duodenal ulcer unless the lesion is located very high.

Final diagnosis:	Carcinoma of stomach.	Peptic ulcer, benign.
Patient:	Woman, aged 41.	Man, aged 60.
History:	Peptic ulcer five years, occasional tarry stools, one large hematemesis in January, 1945.	Peptic ulcer two years. Excision of gastric ulcer elsewhere in February, 1947. Symptoms recurred in three months.
Roentgenologic Examination:	Stomach normal.	Annular defect in pars media with large ulcer crater along lesser curvature. 30 per cent gastric retention in six hours. Impression: gastric neoplasm.
Gastroscopic Examination:	Stomach normal.	None.
Laboratory Examination:	Gastric analysis: Free 24 Total 40 Hemoglobin 8 Gm. (Recent hemorrhage)	Gastric analysis: Free 48 Total 60 Hemoglobin 13 Gm.
Preoperative Diagnosis:	Peptic ulcer.	Carcinoma of stomach.
Operation and Pathologic Examination:	Sept. 22, 1947. Excision shallow ulcer 0.5 cm. in diameter approximately 2.5 cm. below cardioesophageal junction on lesser curvature. Ulcer apparently healing. Impression of surgeon: healing peptic ulcer. Frozen section: no evidence of malignancy. Bilateral vagus resection and gastroenterostomy. Permanent section from another portion of ulcer; adenocarcinoma, poorly differentiated, arising in chronic peptic ulcer (fig. 1). Sept. 29, 1947: Wide resection of area of previous excision of gastric ulcer.	Nov. 17, 1947. Subtotal gastric resection. Impression of surgeon: carcinoma of stomach. Gross specimen: ulcer 6 x 4.5 cm. with crater 1.5 cm. deep, dumbbell-shaped and annular 6.5 cm. above pylorus. Chronic peptic ulcer with no evidence of malignancy. Frozen section and permanent sections confirmed benign nature of lesion (fig. 2).

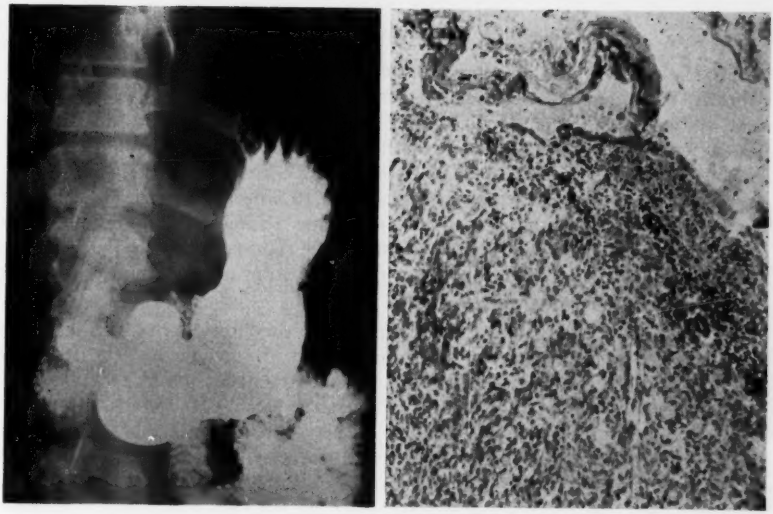

FIG. 1. Adenocarcinoma of the stomach apparently arising in a chronic peptic ulcer.

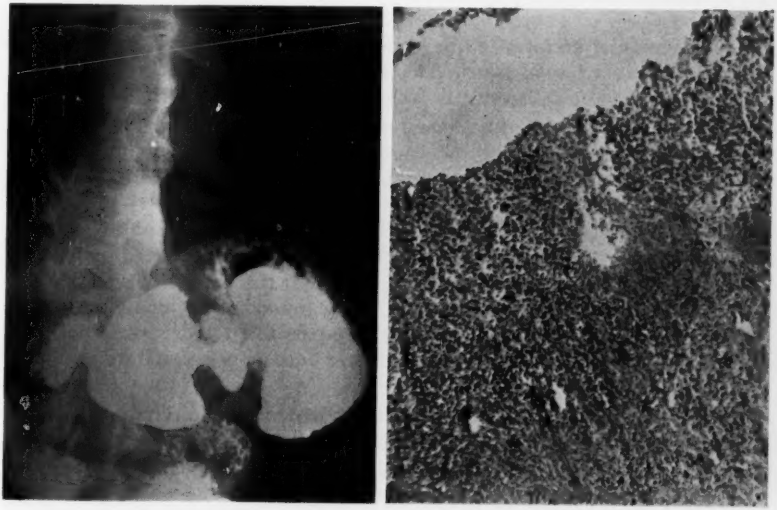

FIG. 2. Chronic peptic ulcer of stomach showing no evidence of malignancy.

Indications for Operation for Gastric Ulcer

Since gastric ulcer cannot be certainly differentiated from gastric carcinoma either by roentgenologic examination, by gastroscopy, or by analysis of clinical data, its treatment is primarily a surgical problem. By this it is not implied that all gastric ulcers should be resected as soon as the diagnosis is made and without a trial on medical therapy. But large gastric ulcers, recurrent gastric ulcers, and gastric ulcers which persist in spite of an adequate trial of a month of medical treatment should be resected unless strong contraindications are present. The criteria of healing are failure of the roentgenologic or gastroscopic examination to demonstrate an ulcer, complete subsidence of symptoms, and disappearance of blood from the stool.

Because gastric resection gives maximum protection against the development of carcinoma or against its recurrence in the event that the lesion is malignant, gastric resection is the preferred treatment for most cases of gastric ulcer. In a small series of patients with gastric ulcer who were subjected to vagotomy without excision of the ulcer the results have been only fair. Two of the 14 patients have had persistence or recurrence of symptoms, and in these cases the possibility of malignancy cannot be excluded. Vagotomy is not recommended unless the ulcer is excised and examined microscopically.

If the ulcer is on the lesser curvature it cannot be excised without disturbing the motility of the stomach. The shortening of the lesser curvature or the disturbance of the peristaltic wave or a combination of these factors may interfere with the emptying of the stomach and cause retention. Vagotomy increases this tendency. Therefore, if an ulcer on the lesser curvature is treated by excision and vagotomy, a gastroenterostomy also should be made. But the combination of vagotomy, excision of the ulcer, and gastroenterostomy is about as formidable a procedure as gastric resection and is attended by no better results. For this reason gastric resection is preferable.

If the ulcer is small and is located on either the anterior or posterior wall, it can be treated by excision and vagotomy without gastroenterostomy. The results in these cases are good, but the number of ulcers adaptable to this treatment is small.

If the ulcer is so high on the lesser curvature that nothing short of a total or subtotal gastrectomy could effectively remove it, or in cases in which the technical difficulties of resection are so great that the danger of resection outweighs the danger that the lesion may be a carcinoma, vagotomy alone, preferably accompanied by a biopsy, may be preferable to the risk of resection. Again these cases are not common and afford only an occasional indication for the treatment of gastric ulcer by vagotomy.

Biopsy of gastric ulcers is not a satisfactory method of excluding the possibility of carcinoma. Excision is much more certain but is not always practicable. In those cases in which technical difficulties render resection or excision undesirable, a biopsy can be taken with a biopsy forceps introduced through a small gastrotomy or, in the event that a gastroenterostomy is made, through the stoma. Specimens may be obtained even from the highest lesions in this man-

ner. Two or three specimens from different parts of the margin of the ulcer should be taken because it is not always possible for the pathologist to determine the presence of malignancy on the basis of a single biopsy. Furthermore, as in the case reported, one part of the ulcer may be benign and another part malignant. Since these difficulties are avoided by gastric resection and since the results of resection are so good, it should be employed as the preferred treatment except in special cases.

Summary

1. It is not always possible to distinguish between peptic ulcer and carcinoma of the stomach.

2. Most cases of gastric ulcer requiring operation should be treated by gastric resection rather than by vagotomy.

3. Vagotomy for gastric ulcer is not recommended unless (a) the ulcer is excised and examined microscopically or (b) the ulcer is so high that it cannot be resected without performing a total gastrectomy or incurring undue risk.

CARCINOMA OF THE STOMACH

Two "Atypical Cases"

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CARCINOMA of the stomach may escape detection even when all modern aids to diagnosis are readily available. The occasional appearance of obscure cases in clinical practice should make physicians alert to the possibility of gastric cancer in patients who may not manifest the usual gastric symptoms.

When the tumor involves the lower esophageal or pyloric orifice it is common for symptoms to develop relatively early. In involvement of the esophagus there may be pain or substernal distress during or shortly after eating, associated with a sense of fullness in the upper abdomen, as well as belching or anorexia. In the pyloric lesion symptoms of gastric retention appear fairly early in the disease process and are manifested by a sense of pressure, anorexia, and vomiting or eructation of food, often bile-stained and containing red or coffee colored blood.

The greatest problem in the diagnosis of gastric cancer presents itself when the tumor involves the cardia without extension to the esophagus or when the greater curvature of the stomach is the site of malignant change. As long as the magenstrasse permits a free flow of food from the esophagus to the duodenum the real cause of the patient's symptoms is sometimes overlooked. Such an error is especially apt to occur when the patient is well nourished, has a good appetite, has insignificant indigestion or none at all, and when a mass cannot be palpated on abdominal examination.

When the patient is under the age of forty and the symptoms are limited to moderate anorexia, fatigue, fever, weight loss in keeping with the degree of appetite loss, and other symptoms suggesting a generalized disease process, causes other than malignant disease are frequently suspected. It is often thought that the difficulty may be due to blood dyscrasias, bacteremia, tuberculosis, disseminated lupus erythematosis, arteriosclerosis, or rheumatic diesase. An error in the primary diagnosis is enhanced by the finding of a moderate leukocytosis without anemia, a high sedimentation rate, fever, or a history that points to disease processes elsewhere than in the stomach.

In the following 2 cases of gastric carcinoma the correct diagnosis was made only after an extensive period of observation and study.

Case Reports

Case 1. A woman, aged 30, and mother of one child, was admitted to the Cleveland Clinic on March 10, 1930. She complained of pronounced fatigue, generalized aching in the back and legs and a troublesome bitemporal headache. Manifestations of a gastric disorder

were meager and consisted of poor appetite and eructations of gas of a foul odor. There was no indigestion, vomiting, diarrhea or pain.

The symptoms began about two weeks before admission and for a period of three days the patient's temperature had reached a maximum of 101 F.; previous to this time she was not aware of fever.

Two and one-half years before the patient had experienced a mild dyspepsia of some months duration. However, roentgenologic studies of the gastrointestinal tract at that time were reported to have been normal. The gnawing hunger pains which accompanied this condition disappeared in a few weeks following the use of extra milk between meals. She remained free of gastric symptoms for over two years.

History of past illnesses did not reveal any significant facts pertaining to her present condition. However, six months before admission she had moved to a farm where she drank unpasteurized milk, but during this period manifested no symptoms of acute brucellosis and gained 18 pounds.

Physical examination did not reveal abnormalities except a pulse rate of 120 and a temperature 103.2 F. The breath had an unusually foul odor suggesting atrophic rhinitis although this condition was absent and no foci of infection were noted in the mouth or throat. No enlargement of lymph glands or evidence of disease in the lungs, heart, rectum, pelvis, or extremities was observed. There were no signs of phlebitis, perirenal tenderness or arthritis. There was no abdominal distention, fluid, masses, or tenderness; the liver, spleen and kidneys were not palpable.

The clinical impression was chronic brucellosis. The blood counts showed 3,480,000 red blood cells; 11,350 white cells; the hemoglobin was 9.5 Gm. The differential count showed polymorphonuclears 88, lymphocytes 7, eosinophils 1, and monocytes 4. Blood culture was sterile. Agglutination tests for undulant fever, typhoid, paratyphoid A and B, proteus O-x19 were all negative. Chest roentgenogram was normal, urine culture was sterile and blood sugar, blood Wassermann and Kahn tests, and sedimentation rate were all within normal limits.

During a period of eighteen days in the hospital the temperature varied between 103 degrees and normal; later after exploratory operation it remained at about 100 degrees. Roentgenologic studies with cholecystographic dye showed a normal functioning gallbladder without stones. A barium meal entered the stomach without delay but a large infiltrative lesion involving the cardia and body of the stomach was found in pyloric emptying. The small bowel studies did not reveal any abnormalities.

Neither tumor at the lower end of the esophagus nor obstruction at the cardia were found by esophagoscopy. Since the lower esophagus was not involved in the tumor process and no evidence for metastasis was found in the liver and lungs operation for total removal of the stomach was considered advisable. Surgical exploration was performed by Dr. T. E. Jones on March 22, 1948. The lesion was extremely large, involving most of the stomach with extension to adjacent lymph nodes and spleen. Since the tumor was inoperable the incision was closed without resection of the stomach.

Comment

Among the unusual features of this case were the youth of the patient (30 years), the short duration of symptoms (two weeks), the absence of symptoms of gastric disease except eructation of foul gas and the general appearance of a chronic infectious process. Although the sedimentation rate was normal on two occasions there was moderate anemia and leukocytosis. However, agglutination tests, bacterial culture studies of the urine and blood, and roentgenograms of the chest were normal.

Gastrointestinal roentgenologic studies were made principally because of the unexplained fever and fetid odor of the breath. In the absence of pathologic

changes in the mouth and upper respiratory tract and of cough it was thought advisable to make a thorough study of the gastrointestinal tract.

Case 2. A man, aged 40, was first admitted to the Cleveland Clinic on the orthopedic service on November 8, 1944. At that time he had a chronic synovitis of the left ankle. Operation was performed by Dr. James A. Dickson after x-ray of the ankle showed no bone changes. A granulomatous mass was removed which on pathologic examination showed tissue containing giant cells consistent with a diagnosis of tuberculous granuloma. Tubercle bacilli were not seen on stained specimens. X-ray of lungs showed no pulmonary tuberculosis. The wound healed by first intention.

This patient entered the Cleveland Clinic on March 5, 1948, with the history of fatigue, weight loss of 18 pounds in the past three months, and loss of appetite. There was no history of fever, chills, night sweats, or cough. He complained of a dull ache in the lower back which would awaken him at night and pain in the left flank on sneezing or coughing.

The gastric symptoms consisted of eructation of gas and rare heart burn, but no colic and no jaundice. The bowels were regular and melena was not noted by patient. There were no genitourinary symptoms.

Past history was not significant except that the patient was in a tuberculosis sanatorium for sixteen months in 1931, but his sputum never showed tubercle bacilli and roentgenograms of the lung did not reveal any abnormalities.

Physical examination of the abdomen showed nothing unusual except a mass in the left upper quadrant which was readily palpable 2 fingers' breadth below the costal margin, was firm with a sharp edge, moved with respiration, and was thought to be an enlarged spleen.

Rectal study showed a tender, firm mass in the rectal pouch, which was interpreted to be tuberculosis of the left seminal vesicle although the possibility of a Blumer's shelf was considered. The extremities and joints were normal save for some fixation of left ankle due to a previous operation.

The urine showed 15 to 20 white blood cells per high power field with an occasional red blood cell. An intravenous urogram demonstrated prompt excretion from both kidneys with a minimal hydronephrosis and a one-hour delay of emptying of the left renal pelvis. There was slight narrowing of the ureter just distal to renal pelvis on the left. Cystoscopic examination revealed mild cystitis; bilateral retrograde pyelograms were normal.

The red blood cell count was 3,930,000, hemoglobin 10.5 Gm., white blood cells 7350 and sedimentation rate was three times the upper limit of normal. Blood sugar was normal; Wassermann and Kahn tests were negative. X-ray of the chest showed a small area of density at the left costophrenic angle thought to be fluid.

On the patient's third admission to the Clinic March 24, 1948, the symptoms were similar to those present previously with the addition of abdominal pain of a generalized burning nature, made worse by eating but relieved temporarily by belching.

The abdomen was distended, tender throughout and considerable ascites present were shown by a fluid wave on percussion. Rectal study showed the mass in the rectal pouch to be larger, fixed, and tender. There was no epididymitis and intradermal tests for tuberculosis and histoplasmosis were negative. At this time a second retrograde pyelogram showed some dilation of the calyces of the left kidney. Abdominal paracentesis revealed a cloudy amber fluid, 150 cells per cc. chiefly lymphocytes and many large bodies suggestive of neoplastic cells. Tumor cells were not found.

Inasmuch as we considered the ascites to be due to tuberculous peritonitis a peritoneoscopy was performed by Dr. George Crile, Jr. There was no sign of peritonitis or of enlargement of the liver or spleen. Pathologic study of a large specimen of ascitic fluid showed typical malignant cells.

Pathologic study of the sediment obtained by centrifugation revealed sheets of cells with hyperchromic irregular nuclei and pale, well defined cytoplasm. Tumor giant cells and mitoses were rather frequently seen. The pathologic diagnosis was "neoplastic cells present, probably carcinoma."

Comment

This patient received a long and somewhat involved diagnostic study. It appeared obvious that he was suffering from generalized tuberculosis. Three and one-half years ago pathologic study revealed tuberculous synovitis of an ankle. His left ureter was narrowed by a stricture, tuberculous seminal vesiculitis was suspected and he had developed ascites. No pulmonary disease, malignant or otherwise, was disclosed by roentgen study. Furthermore peritoneoscopy showed no signs of a tuberculous or inflammatory process and the tuberculin test was negative.

With the discovery of tumor cells in the ascitic fluid and the presence of an obstructing tumor of the distal half of the stomach the correct diagnosis was made.

Conclusion

Cancer of the stomach may cause fever, leukocytosis and an elevated blood sedimentation rate. When such a lesion is masked by symptoms suggesting a generalized disease or infectious process it is important to search for objective facts chiefly by means of laboratory tests. By careful analysis of such findings the true diagnosis of carcinoma can be made in spite of atypical symptoms.

THE FRANK E. BUNTS EDUCATIONAL INSTITUTE

A continuation course will be presented on
Thursday, Friday, and Saturday, October 7, 8, and 9, 1948

DISEASES OF THE DIGESTIVE TRACT

Tentative Program

Thursday, October 7, 1948

8:00-9:00 a.m.	Registration	
9:00 a.m.	Dysphagia: Diagnosis and Medical Management	E. N. Collins, M.D.
9:20 a.m.	Esophagoscopy: Treatment of Lesions in Esophagus	H. E. Harris, M.D.
9:40 a.m.	Surgical Treatment of Esophageal Diseases	George Crile, Jr., M.D.
10:00 a.m.	Pancreatic Tumors and Other Pathologic Disorders of Upper Digestive Tract	J. B. Hazard, M.D.
10:20 a.m.	Roentgen Examination of the Esophagus and Stomach	John R. Hannan, M.D.
10:40 a.m.	Gastroscopy	H. R. Rossmiller, M.D.
11:00 a.m.	Neoplasms of the Stomach	R. R. Bartunek, M.D.
11:20 a.m.	Peptic Ulcer: Complications and Medical Management	E. N. Collins, M.D.
1:00 p.m.	Exhibits: Model Demonstrations of Esophagoscope and Gastroscope	
2:00 p.m.	Surgical Treatment of Gastric and Duodenal Diseases	George Crile, Jr., M.D.
2:30 p.m.	Roentgen Examinations of Liver, Biliary Tract, and Pancreas	C. R. Hughes, M.D.
3:00 p.m.	Present Status of Liver Function Tests	Charles Brown, M.D.
3:30 p.m.	Differential Diagnosis and Treatment of Jaundice; Liver Biopsy, Laennec's Cirrhosis, and Portal Hypertension Syndrome	H. R. Rossmiller, M.D.
4:30 p.m.	Surgical Treatment of Cholecystitis	R. S. Dinsmore, M.D.
	GUEST SPEAKER	
8:00 p.m.	Bilateral Vagotomy in Treatment of Peptic Ulcer	Lester R. Dragstedt, M.D. Professor of Surgery, University of Chicago

Friday, October 8, 1948

9:00 a.m.	Non-tropical Sprue	Charles Brown, M.D.
9:30 a.m.	Diseases of the Pancreas	R. R. Bartunek, M.D.
10:00 a.m.	Splanchnic Block	D. E. Hale, M.D.
10:30 a.m.	Surgery of the Pancreas	George Crile, Jr., M.D.
11:00 a.m.	Gastric Neuroses	L. J. Karnosh, M.D.
11:30 a.m.	Gastrointestinal Symptoms Due to Genitourinary Lesions	W. J. Engel, M.D.

Meals can be obtained in the cafeteria of the Cleveland Clinic Hospital.

1:00 p.m. . . . . Exhibits and Demonstrations: Stool Examinations, Cultures, Blood Agglutination for Dysentery, etc. . . . . .
2:00 p.m. . . . . Acute Abdominal Diseases; Appendicitis . . . R. S. DINSMORE, M.D.
2:30 p.m. . . . . Regional Enteritis . . . . . H. R. ROSSMILLER, M.D.
3:00 p.m. . . . . Roentgen Examination of the Intestines . . . . . J. C. ROOT, M.D.
3:30 p.m. . . . . Diagnosis and Medical Management of Colonic Disorders . . . . . E. N. COLLINS, M.D.
4:30 p.m. . . . . Surgical Treatment of Diseases of the Colon and Rectum . . . . . T. E. JONES, M.D.

5:00 p.m. . . . . Cocktail Hour, Hotel Bolton . . . . . .

Saturday, October 9, 1948

9:00 a.m. . . . . Pathology of Diseases of the Intestines . . . . . J. B. HAZARD, M.D.

GUEST SPEAKER

9:30 a.m. . . . . Intestinal Intubation—Physiologic and Clinical Aspects . . . . . T. GRIER MILLER, M.D.
Professor of Clinical Medicine, University of Pennsylvania

10:15 a.m. . . . . Panel Discussion—Questions and Answers
Drs. Miller, Crile, Collins, Jones, and Rossmiller with Dr. Jordan as Moderator.

Questions pertaining to disorders of the digestive system may be submitted for the panel discussion on Saturday morning. A "Question Box" during the first two days of the course will be available for the convenience of those wishing to present questions to the members of the panel.

REGISTRATION BLANK

THE FRANK E. BUNTS EDUCATIONAL INSTITUTE . . . . . ., 1948
Cleveland Clinic Building
2020 East Ninety-third Street
Cleveland 6, Ohio

Gentlemen:

Please register me for the course on "Diseases of the Digestive Tract" which is to be given October 7, 8, and 9, 1948.

I am sending check for $5.00, and the remainder of the fee, $5.00, will be paid on Registration, October 7.

NOTE: Checks should be made payable to the Frank E. Bunts Institute. Mail registration blank and check to Dr. Edwin P. Jordan, Cleveland Clinic Building, 2020 East Ninety-third Street, Cleveland 6, Ohio.

Name .....

Address .....

Medical School and
date of graduation .....

This course is open only to graduates of approved medical schools. Registration will be limited to the number of physicians who can be adequately accommodated.

CLEVELAND CLINIC NEWS

APPOINTMENTS

Dr. J. R. Haserick of Minneapolis has been appointed to the Staff in the Department of Dermatology. Following graduation from the University of Minnesota Medical School in 1941, Dr. Haserick served a year of Fellowship in Medicine subsequently entering the Army where he spent two and one-half years. He completed two more years of work in dermatology at the University of Minnesota following his separation from the Army.

Dr. Haserick comes to the Cleveland Clinic from the University of Minnesota where he has been an instructor in the Department of Dermatology. He is certified by the American Board of Dermatology.

Dr. John D. Battle has been made a member of the Staff in the Division of Internal Medicine. Dr. Battle graduated from Washington and Lee University in 1934, and received his M.D. degree from the University of Pennsylvania in 1938.

Dr. Battle was a Fellow at the Cleveland Clinic from 1940 to 1942. He is certified by the American Board of Internal Medicine, served four years in the U. S. Navy, and has written several articles for the medical literature.

BUILDING PROGRESS

The new seven-story addition to the Clinic building is practically completed, and the departments have begun to move into their new quarters. The entire fifth floor is devoted to the clinical laboratories. As soon as possible the lower three floors will be remodeled to match the new sections. The second floor is to be devoted entirely to the Department of Roentgenology.

LOWER THESIS PRIZE

The 1948 Lower Fellowship Thesis Prize was awarded on June 15 to Dr. Alys Lipscomb for her paper entitled "Incidence of Various Types of Essential Hypertension as Related to Evaluation of Successful Therapy." The following additional theses were awarded honorable mention: Dr. E. R. Kyger, Jr., "Cutaneous Brucella Sensitivity in Multiple Sclerosis," Dr. J. J. Reinhard, "Hemorrhagic Shock in Hepatectomized and Bilaterally Nephrectomized, Hepatectomized Dogs," and Dr. E. C. Weiford, "Tantalum Cranioplasty."

THE CLEVELAND CLINIC FOUNDATION

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